Editorial

Deleterious Compounds in Foods

The progress of medical and nutritional science over the past century has armed the physician with knowledge and tools with which to combat infectious and deficiency diseases of man. Although people in many parts of the world still suffer from parasitism, infections and the lack of essential nutrients, these troubles are now primarily the results of political, religious and economic, rather than scientific, matters. The essential facts are largely known and need only to be applied.

On the other hand it is becoming increasingly clear that a great deal more still needs to be learned about substances in ordinary foods which, although not acutely toxic, nevertheless may be capable of producing or contributing to the development of diseased conditions when consumed over long periods of time.

Some food-related illnesses are, of course, well known—food poisoning, ergotism, various allergies and the like. Some foods are known to be on the border line of safety but are eaten anyway by certain groups either because of preference or dire need. Lathyrism, favism and paralytic shellfish poisoning are examples. Another may be poisoning from the cycad nut which is consumed as a major source of starch in many areas of the Pacific even though it is known to contain toxic chemicals which are more or less completely removed before use by soaking the nuts in water.

Other foods (or feeds) may be undesirable because they contain chemicals which bring about the indirect development of a deficiency of some essential nutrient. Thus, linseed contains an antagonist of vitamin B_6, and it has been suggested, although not established, that pellagra may be caused by the presence of an antiniacin factor in corn rather than by a deficiency of nicotinic acid and/or tryptophan.

The main concern of this note, however, is with the compounds present in ordinary "good" foods which may have undesirable effects over an extended period of time. Probably the best known examples at present involve the possible relationship of dietary fats to atherosclerosis, soluble carbohydrates to dental caries and goitrogenic substances (in certain vegetables and in the milk of cows grazing on certain weeds) to endemic goiter. In addition to these fairly well known and extensively discussed situations, there are probably many more similar instances in which the basic facts are even less well understood.

One interesting example, which has received attention during the past several years, is the effect produced by feeding flowering sweet pea (*Lathyrus odoratus*) seeds to young rats, chicks or turkey poults. This results in extensive damage to mesenchymal tissues, manifested by such conditions as bone deformities, curvature of the spine, hernias and dissecting aneurysms of the aorta. The causative agent, identified in 1954 as β-aminopropionitrile (BAPN) $NH_2CH_2CH_2CN$, occurs in the form of a conjugate with glutamic acid which is enzymatically hydrolyzed *in vivo* to release the active toxin. Oxidation, presumably under the influence of monamine oxidase, also occurs *in vivo* with the formation of a nontoxic product, cyanoacetic acid, $CNCH_2COOH$. Inhibitors of monamine oxidase such as Marsilid® markedly increase or "potentiate" the toxicity of BAPN. The severity of BAPN poisoning, which might result from ingestion of sweet pea seeds by an animal, would depend, therefore, among other factors, on the amount consumed, age (the young are more susceptible), tissue level of monamine oxidase and of the enzyme which hydrolyzes the conjugate, and on whether or not inhibitors of these enzymes had been

ingested recently. Somewhat similar considerations could well apply to man's intake of physiologically potent amines, such as serotonin, norepinephrine, γ-amino butyric acid, α,γ-diamino butyric acid, and probably many others that may be present in various foods. If these should happen to be ingested simultaneously with some of the monamine oxidase inhibiting drugs, serious damage to connective tissue might well be the result.

Other such interrelationships, which under certain combinations of circumstances could lead to undesirable effects from certain foods, undoubtedly exist. Their recognition is slow and arduous because it is difficult to establish a cause and effect relationship between the consumption of a certain food by human beings and the appearance of a diseased condition many years later. Nevertheless I strongly suspect it is precisely in this area that the science of nutrition has its greatest opportunity to contribute to man's welfare and that the clinician will need to be alert to new developments.

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